Wernicke and Henschen believe the centre to be located in the infra-parieta lobule. Wernicke analyzed published reported cases and concludes :

I. Conjugate deviation of the eyes is always in relation with a lesion of the infra-parietal lobule or of fibres emanating from it.

2. Lesions of the infra-parietal lobule always produce conjugate deviation of the eyes, if only transitory.

Charcot and Pitres believe the following invalidates Wernicke's conclusions :

1. We have reported cases where lesions of the infra-parietal lobule were not accompanied with conjugate deviation of the eyes.

2. Conjugate deviation of head and eyes may exist without lesion of infra-parietal lobe A case is cited—Right hemiplegia, conjugate deviated eyes toward left. Circumscribed softening of left ascending convolution alone.

(*d*) *Centre for elevating the superior lid.*

Exceptionally in hemiplegia is found a paralysis of the superior eyelid. Grasset and Landouzy place a cortical centre for same in the angular gyrus, and offer a number of corroboratory observations :

1. Softening of the left angular gyrus—incomplete right ptosis.

2. Softening of left angular gyrus—persistent ptosis right superior lid.

3. Softening of left infra-parietal lobule— right ptosis.

Charcot and Pitres hesitate to admit a definitely localized centre on following grounds :

1. Large number of observations where no ptosis existed, yet autopsies showed extensive alterations of the angular gyrus.

2. Ptosis is described in a large number of cases where the lesions reach beyond, and often far beyond, the region of the angular gyrus.

We saw it exist in two cases, with cortical softening of the inferior extremity of the ascending convolutions. In a third case, with softening of ascending frontal and base of second frontal.

II.

DISTURBANCES OF SENSIBILITY IN THEIR RELATION WITH LESIONS OF THE CORTICAL MOTOR ZONE.

Hitzig offered the hypothesis that troubles of motility caused by destruction of the cortex may be due to perturbations of muscular consciousness.

This hypothesis was accepted with modifications by a number of physiologists—Notnagel, Schiff, Munk, etc.

Notnagel explained motor paralysis of cortical origin by perturbations of muscular sense.

Schiff believed lesions of zone called motor caused tactile anæsthesias, and these, secondarily, ataxias of motion rather than real motor paralysis.

Munk generalizes. He thinks that the brain has no direct action upon motility; the motor region is the region where end and are elaborated the sensations of contact, pressure, temperature, position, etc., of various parts of the body. If lesions here cause perturbations of motion, it is simply because they cause total or partial loss of kinesthetic sensations, which excite and direct motility.

Bastian agrees, denying existence of any motor centres. He thinks the Rolandic area is formed by an aggregation of sensory centres, the activity of which excite voluntary motor centres in pons, medulla and spinal cord.

Ferrier, Bechterew, Horsley maintain, on the other hand, the doctrine of the anatomical and functional separation of motor and sensory centres. They affirm that cerebral mutilations limited to the Rolandic area do not cause *per se* any sensory perturbation.

Clinicians do not agree any more than physiologists. Tripier, Ballet, Exner, Starr and others who have noted frequent co-existence of sensory perturbation with motor paralysis of cortical origin, are inclined to admit hypothesis of superposition of motor and sensory areas.

Inversely, other observers, having found sensibility intact in a large number of cases of hemiplegia dependent on lesions limited to Rolandic convolutions, do not accept theoretical deductions. What are the facts?

Ferrier cites 284 cases: Sensibility not mentioned, 100; sensibility intact, 121; sensibility altered, 63. Motor paralysis of cortical origin exists two out of three times with no sensory perturbation.

In twelve observations brachio-facial monoplegia we have found: Sensibility intact, four; sensibility altered, three; sensibility not mentioned, five.

In twenty-two cases brachio crural monoplegia: Sensibility not mentioned, thirteen; sensibility intact, seven; sensibility altered, two.

In twelve observations, crural monoplegia: Sensibility not mentioned, five; sensibility intact, five; sensibility slightly altered, one; diminished tactile sensibility, entire body with preserved pain and temperature sense, one.

Finally, eight observations, facial monoplegia: Sensibility not mentioned, four; sensibility intact, two; sensibility altered, two.

In one of the latter two cases anæsthesia extends beyond face to upper part right side of trunk; in the other case it was general.

Sensory phenomena not only are not superimposed upon motor, but they are transient. All who have studied them agree upon this. Shall we say they are capable of displacing themselves from one moment to another, of disappearing and reappearing from slight causes if they depend on the destruction of corresponding perceptive centres?

It remains to explain why alteration of motor zone in about one-third cases are accompanied with disturbances, cutaneous or muscular sensibility. Legroux and de Brun refer them to circulatory disturbances which are but beyond points in which paralytic lesion is located.

In our opinion the anæsthesias which sometimes accompanies the motor paralysis of cortical origin are mostly functional, analogous, if not identical, with hysterical anæsthesias—are superadded phenomena, accidental, not depending upon the lesion of the Rolandic region and playing no pathological part in the production of the paralytic symptoms.

III.

LIMITED ATROPHIES OF THE MOTOR ZONE CONSECUTIVE TO OLD AMPUTATIONS OF THE EXTREMITIES.

We know that long after the amputation of an extremity there results a more or less complete atrophy of the corresponding spinal cord segment.

Does atrophy occur in the brain tissue?

Dickson found no atrophy of the brain in four subjects where amputation had been performed, from two to fifty-three years before death.

Vulpian was not more successful.

Later observers reported changes, but many observations are valueless since the amputation followed congenital deformities and acquired atrophies of the limbs. To be of any value we must limit our study to cases in which cerebral atrophy, if it exists, can be traced to loss of limb.

Thirty-seven cases of old amputations are cited: Atrophy more or less marked in motor zone of opposite side in twenty-two cases; unchanged in fifteen cases.

Analyses of these cases does not make it clear why atrophy in some and none in others. The analysis seems to demonstrate that the